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What are adolescent antecedents to antisocial personality disorder?

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ABSTRACT

Background *This paper fills a gap because there are very few studies that prospectively predict antisocial personality disorder (APD) from psychopathology earlier in life in clinic-referred samples of young males.*

Method *The paper addresses the continuity between conduct disorder (CD) and other forms of psychopathology during ages 13–17 and modified APD at ages 18 and 19 (modified to remove the DSM-IV requirement of pre-existing CD by age 15) in the Developmental Trends Study.*

Results *The results show that 82–90% of APD cases met criteria for CD at least once during ages 13–17, and very few youths who met criteria for ODD during this period progressed to APD without intermediate CD. While CD is a strong predictor of modified APD, when other factors were accounted for in regression analyses, the best predictors were callous/unemotional behaviour, depression and marijuana use. ADHD during ages 13–17 was not significant in the final model. Males with CD during adolescence who progressed to APD tended to commit more violence, as evident from their court records.*

Conclusions *Implications are discussed for the conceptualization of developmental models leading to APD, the strengthening of relevant symptoms of CD predictive of APD, and preventive and remedial interventions.*

Introduction

Antisocial personality (APD) is one of the most impairing and socially disastrous disorders during adulthood and is often seen as highly refractive to treatment (Loeber et al., in press). APD clearly arises from childhood conduct problems (Robins, 1966), yet there is little prospective research specifying the characteristics of those children who will later develop APD. It is anticipated that clarification of the long-term antecedents and causes of APD is a first step towards specification of preventive interventions.

Research on the precursors of APD has been held back by several limitations of research to date. First, the definition of APD requires the presence of conduct disorder (CD) prior to adulthood (American Psychiatric Association, 1994). This usually makes it impossible to ascertain to what extent CD is a precursor to APD, unless a modified definition of APD is used that does not include this clause. Those studies that used modified APD have shown that only about 30–40% of children with CD develop APD (Robins, 1966, 1991; Robins et al., 1991). However, it is far from clear what the characteristics are that distinguish those children with CD who developed APD from children with CD who did not develop APD. A second limitation of studies is that a proportion of children outgrow CD between childhood and adolescence. These children constitute 'false positives' in the prediction of APD. For that reason, precursors to APD may be best studied in a period after some of the CD youth typically have desisted from CD. Third, we do not know of any studies that tested whether other disruptive behaviour disorders, such as oppositional defiant disorder (ODD) in the absence of CD, or ADHD with or without CD, constitute precursors to APD.

Loeber et al. (in press) have proposed a hierarchical model of development of APD in males, in which ODD is a necessary precursor to CD, which in turn is a necessary precursor to APD. The hierarchical model can be contrasted with a *de novo* model in which APD emerges with these pre-existing conditions, or in which ODD increases the risk of APD without the intermediate CD. Loeber et al.'s (in press) hierarchical model stipulates that, although a relatively large proportion of boys experience the onset of ODD earlier in life, from this group only a proportion (particularly those who have ODD and who physically fight) escalate to conduct problems and eventually qualify for CD. Out of the latter group, only a subgroup of those with CD will further progress to APD.

A crucial question remains, however, regarding the proportion of boys with CD who develop APD. Robins (1978), summarizing her studies, showed that only about a third of the youth with CD later developed APD. The percentage was somewhat higher (40%) in an English sample of boys who had been reared away from home (Zoccolillo et al., 1992). We reviewed existing retrospective and prospective studies of the prediction of adult APD from childhood CD. We were able to calculate the relative risk of APD in individuals with and without childhood CD from five independent studies (Harrington et al., 1991; Robins, 1966; Robins and Ratcliff, 1979; Robins et al., 1991; Zoccolillo et al., 1992). The relative risks ranged from 3.2 to 18.0, and when combined in meta-analytic fashion across differing definitions of disorder and design, but weighting for sample size, the overall estimate of relative risk was 16.8 for the prediction of CD from APD. Specifically, 1.7% of individuals without a history of CD were given the diagnosis of APD in adulthood across studies compared with 28.5% of individuals with a history of childhood of CD. This very high relative risk for predicting APD from childhood CD is

consistent with the DSM-IV view that few individuals will meet adult criteria for APD without exhibiting at least three symptoms of CD in childhood.

Against this background of findings on the continuity of disruptive behaviour over time, we should consider two other important aspects that remain to be investigated. Previous studies have rarely addressed the 'true' continuity between disruptive behaviours (Lahey et al., 1995; Loeber, 1991). Continuity often occurs against the background of short-term fluctuations in the severity of the disorder. This is often lost in empirical studies limited to only two assessments. In our own work in the DTS (Lahey et al., 1995), we found that in many boys the number of CD symptoms fluctuated above and below the diagnostic threshold from year to year, but remained relatively high. Among boys with CD in Year 1, 88% met criteria again for CD in one or more of the next three assessment years, and 54% met criteria again for CD in two or more subsequent years. Thus, the continuity of CD can be seen to be substantially greater when more frequent assessments are conducted, because a substantial proportion of boys who did not qualify for CD at one follow-up assessment do so in one or more subsequent assessments. We expect that this also would apply to the assessment of APD.

The DSM-IV requirement of APD being diagnosed in adulthood only (American Psychiatric Association, 1994) ignores the possibility that some relevant symptoms of psychopathology may already be present during childhood and adolescence. This is an important point, because knowledge of the developmental continuity between CD and APD may be enhanced by researchers and clinicians attending to early signs of APD. For example, research by Lynam (1996) has used a construct of early psychopathy, while Frick and colleagues (Frick et al., 1994) focused on a construct called callous/unemotional behaviour.

The conceptualization of APD as a product of earlier continuity of similar behaviours, such as CD and callous/unemotional behaviour, may be too simplistic. Research on adult cases of APD clearly shows that they often have comorbid conditions, such as heavy alcohol and drug use, and depression. The unanswered question is to what extent early manifestations of later comorbid conditions aid in the prediction of APD.

It can be argued that the precursors of APD should be studied longitudinally in population samples. However, given the low base rate of APD in the general population (Robins et al., 1991), this would require very large samples. Studies with such large samples have rarely had the resources to assess all participants by means of psychiatric interviews and repeatedly collect information on potential precursors of APD from childhood through adolescence. For these reasons, it is opportune to study precursors of APD in a high-risk population, such as boys referred for disruptive behaviour during childhood.

This paper addresses the following questions pertaining to modified APD:

- (1) How well do CD and callous/emotional behaviour predict APD?

- (2) Do other forms of psychopathology predict APD as well?
- (3) Which factors distinguish between those who will progress to APD from CD and those who do not?
- (4) What is the outcome of those with CD in adolescence who do not progress to APD?
- (5) Is there something unique about the proportion of those with APD who did not demonstrate prior CD? These questions are addressed in data from the Developmental Trends Study, a longitudinal study on the development of APD from childhood onward in clinic-referred boys. We are not aware of other longitudinal studies that have addressed these questions.

Methods

Details of data collection in the Developmental Trends Study can be found in Loeber and colleagues (2000). This clinic-referred sample of 177 boys was recruited in 1987 from clinics in Pittsburgh, Pennsylvania and in Athens and Atlanta, Georgia. The participants were seven to 12 years of age at the beginning of the study, and were followed up annually with parent and child assessments until the age of 17. At 18 and again at 19, participants completed interviews, but parental report was no longer sought. Additional information was obtained through interviews with teachers during the first four years of the study. However, since the focus of this paper is the period of adolescence to young adulthood, and since teacher data come largely from the childhood period, it is not used in the present study.

Clinical measures

Each assessment included a structured clinical interview. When participants were between the ages of seven and 17, the NIMH Diagnostic Interview Schedule for Children (DISC-C; Costello et al., 1982), and a parallel version for parents (DISC-P) were used to assess conduct disorder (CD), oppositional defiant disorder (ODD), attention-deficit/hyperactivity disorder (ADHD), overanxious disorder (OAD), separation anxiety disorder (SAD), dysthymia, and major depressive disorder. Dysthymia and major depressive disorder were combined, using an either/or rule, into depression. Similarly, OAD and SAD were combined into anxiety. We also identified the presence of inattention and hyperactivity-impulsivity dimensions of ADHD (four of six inattention symptoms or six of eight hyperactivity-impulsivity symptoms during any one year). For more detailed description of the identification of these dimensions, see Burke et al. (2001). Teacher DISC reports of child psychopathology were collected during years 1 to 4, and were thus available for use in only the older half of the sample. However, examination revealed that whether or not teacher data were available did not alter the statistical association of the psychopathology variables with APD.

Regarding CD, although the diagnostic interview used DSM-III-R criteria, our aim was to define CD in accord with the criteria established in DSM-IV. The reader is referred to Burke, Loeber, Mutchka and Lahey (this issue) for a discussion of the development of DSM-IV CD within these data. Each diagnosis was dichotomized to indicate whether or not criteria were met for the disorder between the ages of 13 and 17.

At 18 and 19, the Computerized Diagnostic Interview Schedule (Revised) (Robins and Helzer, 1988) was administered to participants. This included an assessment of criteria for APD. Items for APD were structured within the interview for the purposes of assessing DSM-III-R APD. Changes for the DSM-IV criteria for APD included dropping specific items regarding lacking the ability to function as a responsible parent and never having sustained a monogamous relationship for one year. Additionally, two items, pertaining to inconsistent work behaviour and failure to honour financial obligations, were combined. We developed DSM-IV scoring by excluding data on the two items that were dropped, and defining the item on work behaviour and financial obligations by the combined pool of items for these criteria within DSM-III-R. Since we wished to examine CD as an antecedent to APD, we did not employ the requirement that CD had been present to identify APD (called here modified APD). Participants were coded positive for modified APD if they met criteria at either age 18 or age 19.

Substance use

Self-report items regarding substance use in four categories (tobacco, alcohol, marijuana, and other drugs) were included in the assessment battery beginning in the third year of the project (when the participants were nine to 14 years of age). The category of other drug use included tranquillizers, barbiturates, codeine, amphetamines, LSD, cocaine, crack, heroin and PCP. Participants were asked to estimate the number of days out of the past year they used each substance. The reported number of times for each age between 13 and 17 was summed, and dichotomized so that the top quartile of users was coded 1 and the remainder 0. Specific cut points were 730 or more days of tobacco use, 87 or more days of drinking, and 31 or more days of marijuana use. In the case of other drug use, any reported use resulted in that person ranking among the top quartile of users, so this category is in essence any reported other drug use.

Callous and unemotional behaviour

Parents and teachers completed child behaviour checklist forms annually. Items from the CBCL were used to identify callous/unemotional behaviour (Frick et al., 1994), which includes: acts explosively, gets arrested, brags, is daring, is impulsive, acts sneakily, manipulates others, is a smooth talker, and

lacks guilt. We wished to reduce the similarity between these items and our diagnostic variables and to identify a factor of facility with interpersonal manipulation and deception. For that reason, we restricted the callous/unemotional construct to: acts sneakily, manipulates others, is a smooth talker, and lacks guilt. Positive endorsement was defined as a response of true or very true for the item by either the parent or teacher. Items were summed across ages 13 to 17, generating a range from 0 to 20 ($M = 10.81$, $SD = 5.35$).

Adult criminality

Adult criminality was determined by a review of charges listed on state (Pennsylvania and Georgia) and federal (Federal Bureau of Investigation) adult criminal records. We developed two constructs: the total number of charges, and the number of violent charges. Violent charges included homicide, rape, aggravated and other assaults, robbery, battery, rioting, and child abuse. In some cases, charges were reported from one source and not the other. We resolved these cases by including any non-redundant charges from either source.

Analyses

Data used

The focus of this paper is the development from adolescent CD to APD in young adulthood. Several papers have described the early progression within this sample from ODD to CD and the course of CD over the early and middle waves (Lahey et al., 1995; Lahey et al., in press). For these reasons, data used in this paper are based on participants' behaviour between the ages of 13 and 19.

Missing values

Of the original sample of 177, 163 participants completed at least one adult assessment, leaving 14 missing from the modified APD construct. Additionally, cases were coded as missing if they had more than two missing values of the five phases used to create adolescent variables. This resulted in five additional cases being excluded due to missing adolescent data, and a total of 158 cases with valid data across adolescence and young adulthood. We tested missing cases to determine whether any differences existed between those missing and those with valid data. For those missing from the APD variable, only adolescent depression discriminated them from non-missing cases: those with adolescent depression were less likely ($\chi^2(1) = 5.88$, $p = 0.015$, $OR = 0.12$) to be missing from the APD variable in adulthood. No factors were identified that discriminated between missing and non-missing for adolescent constructs.

Analytic strategy

Variables were screened prior to entry into the multivariate logistic regression. The logit distributions of any continuous scaled variables were examined using smoothed scatter plots to test the assumption of linearity within their logits. Univariate analyses employed chi-square tests for significance. In cases where any expected cell size was less than five, Fisher's exact test was used. No variables produced 'zero cells' within the cross-tabulations.

To examine sets of variables for the prediction of APD, logistic regression analyses were used. Several authors have suggested that in the selection of independent variables in regression models, the traditional alpha value of 0.05 often fails to identify important variables (Hosmer and Lemeshow, 1989). To avoid this problem, the authors recommend the adoption of an alpha level of up to 0.25. We adopted an alpha value of 0.15 during the process of selecting variables for model entry.

The variables within each domain were entered into multivariate logistic regressions to identify those that were most strongly associated with the dependent variables. A backwards selection approach, with an elimination criteria of 0.05, and controlling for age, was used to reduce these variable sets. The surviving variables from each domain were entered into the second stage of backwards logistic regression competing with those variables retained from other domains. Within the final model, variables were examined to identify any interactions. In addition, the calibration and discrimination of each model was determined by examining the Hosmer and Lemeshow goodness-of-fit statistics, and the C statistics (Hosmer and Lemeshow, 1989). In all cases, the model adequately fitted the data. We also tested model tolerance and found no instances of problems of collinearity.

Results

Out of the 158 cases with valid adolescent and adult data, 60 participants (38%) met the modified criteria for APD at either age 18 or 19. During adolescence, 94 (59%) participants met criteria for CD by combined parent-child report. CD strongly predicted modified APD. Of those with CD in adolescence ($n = 94$) over half (52.1%; $n = 49$) went on to meet criteria for modified APD ($\chi^2(1) = 19.74$, $p = <0.001$, $OR = 5.25$.), compared with 17.2% ($n = 11$) of those without adolescent CD ($n = 64$) who later met criteria for modified APD.

History of CD among those with APD

Because the DSM definition of APD includes a history of CD prior to age 15, we briefly refer to both lifetime and adolescent CD here. Of the 60 individuals with modified APD, 54 (90%) had a history that included CD at any point

between 7 and 17, and six participants (10%) progressed to APD without ever having CD. When focusing solely on adolescent CD, 49 of 60 had CD in adolescence (81.7%), while almost one out of five did not show CD after age 12 (11 cases, or 18.3%).

Callous/unemotional behaviour

Does callous/unemotional behaviour add to the prediction of modified APD when adolescent CD is taken into account? A logistic regression model with adolescent CD (Wald (1) = 6.55, $p = 0.01$; OR = 3.12) and callous/unemotional behaviour (Wald (1) = 5.24, $p = 0.022$; OR 1.10) showed that each independently contributed to the prediction of modified APD.

Other adolescent predictors

Table 1 shows that in bivariate analyses the following constructs were related to modified APD: ODD, depression, and ADHD, including the hyperactive/impulsive and inattentive subtypes of ADHD, with odds ratios being very similar, ranging from 2.31 to 3.09 (only anxiety did not predict modified APD). In contrast, the association between different forms of substance use and modified APD was stronger, ranging from 4.09 to 7.57.

Table 1: Bivariate associations with modified antisocial personality disorder			
Variable	χ^2	p	OR
Psychopathology			
ODD	7.96	0.005	2.87
Depression	7.71	0.005	2.52
Anxiety	n/s		
ADHD	6.67	0.010	2.64
Hyperactive/impulsive Subtype	11.35	0.001	3.09
Inattentive subtype	4.40	0.036	2.22
Callous/unemotional behaviours	16.74*	0.001	1.16
Substance use			
Tobacco use	19.29	0.000	4.75
Alcohol use	13.47	0.000	4.00
Marijuana use	27.11	0.000	7.40
Other drug use	13.29	0.000	4.06
<i>Note:</i> *Wald statistic presented, which was derived from regression analysis due to callous/unemotional behaviours being continuously measured. ODD = oppositional defiant disorder; ADHD = attention-deficit hyperactivity disorder.			

A backwards regression model of the significant psychopathology predictors, including callous/unemotional behaviours, was conducted to identify the strongest predictors of modified APD. Since we included inattention and hyperactivity-impulsivity, we excluded ADHD from the model. Three variables remained significant in the model: callous/unemotional behaviours (Wald (1) = 5.78, $p = 0.016$; OR = 1.11), CD (Wald (1) = 5.52, $p = 0.019$; OR = 2.93), and depression (Wald (1) = 5.84, $p = 0.016$, OR = 2.49). Including ADHD rather than its subtypes did not alter the variables retained in the model. A backwards regression model testing the four substance use variables resulted in only tobacco use (Wald (1) = 4.10, $p = 0.043$, OR = 2.38) and marijuana use (Wald (1) = 13.02, $p = 0.001$; OR = 5.61) being retained in the model as significant predictors of modified APD.

Final model of predictors

A final model of predictors of modified APD was tested, including callous/unemotional behaviour, CD, depression, tobacco use and marijuana use (see Table 2). Tobacco use and CD were dropped from the model, leaving callous/unemotional behaviour, depression and marijuana use as significant predictors of later modified APD.

Diagnostic pathways of disruptive behaviour disorder

Tracking the development of disruptive behaviour over the course of childhood to young adulthood reveals the persistence of disruptive behaviour within this sample. ODD is strongly predictive of CD, which in turn is a strong predictor of APD (see Figure 1). However, significant questions remain. First, which factors distinguish between those who will progress to APD from CD and those who do not? Second, what is the outcome of those with CD in adolescence who do not progress to APD ($n = 45$)? Finally, is there something unique about the proportion (10%) of those with APD who did not demonstrate prior CD?

Table 2: Final model of predictors of modified antisocial personality disorder			
Variable	Wald	p	OR
Callous/unemotional	12.32	0.000	1.16
Depression	4.42	0.035	2.32
Marijuana use	19.02	0.000	7.04
Note: Adolescent conduct disorder and tobacco use were removed from the model.			

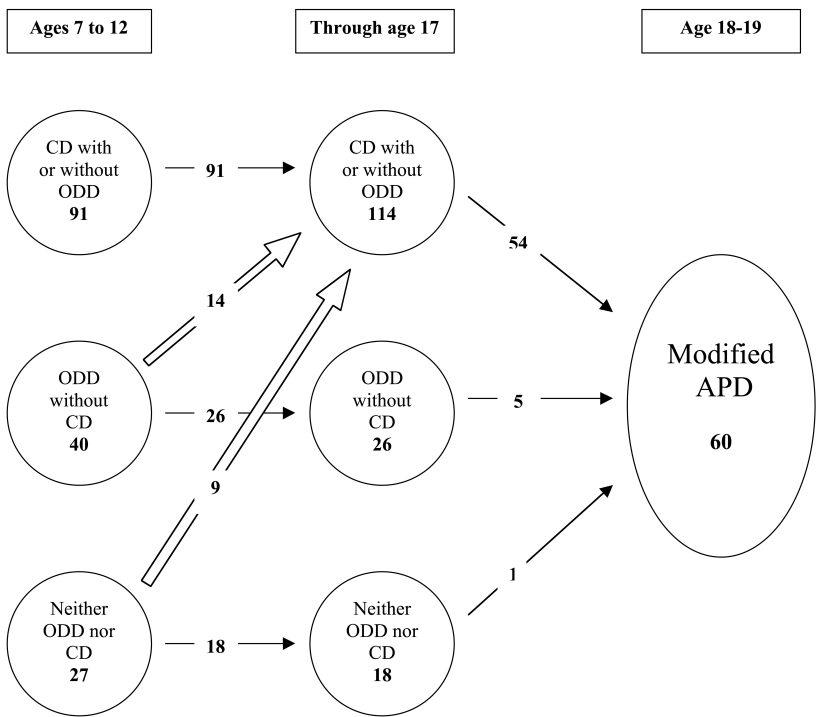


Figure 1: Cumulative history of participants’ oppositional defiant disorder and conduct disorder status in childhood, through adolescence, and progressing into APD in young adulthood.
Note: The number of participants at each point is given within the circles, and the number over-
laying the arrows indicate the number of participants moving between diagnoses between each
period.

Legend:
ODD = oppositional defiant disorder; CD = conduct disorder; APD = antisocial personality disorder.

Because of small numbers, the following results pertaining to these ques-
tions are exploratory. We restricted the data to those who had ever had CD up
to age 17, and coded as 1 those who progressed from CD to APD, and as 0
those who did not (referred to here as continuity). The same set of predictors
tested in the final model of predictors of APD (except for CD), perhaps not
surprisingly, also largely distinguished those who would proceed from CD to
APD from those who did not. The primary difference was that callous/unemo-
tional traits were only marginally significant (see Table 3).

Regarding outcomes of those who do not progress from CD to APD, we
tested the relationship between criminal charges and continuity. Those who
progressed from CD to APD committed more crimes in adulthood than those
who did not ($M = 6.00$, $SD = 6.67$ versus $M = 4.12$, $SD = 6.84$), although this

Table 3: Final model of predictors of continuity between CD and modified antisocial personality disorder			
Variable	Wald	<i>p</i>	OR
Callous/unemotional	4.04	0.044	1.10
Depression	4.84	0.028	2.60
Marijuana use	13.58	0.000	5.87
Note: Tobacco use was removed from the model.			

difference was not statistically significant (Wald (1) = 2.14, *p* = 0.14). However, they also committed more violent crimes (*M* = 0.83, *SD* = 1.40 versus *M* = 0.38, *SD* = 0.99), a difference that was marginally significant (Wald (1) = 3.56, *p* = 0.059, OR = 1.39).

Among the predictors used in these analyses, there were no factors found that distinguished those who progressed from ODD to APD without intermediate CD. The relatively small number of such cases limited our ability to find statistically significant differences.

Discussion

In earlier publications, we demonstrated the hierarchical, predictive relationship between ODD and CD (Loeber et al., 1995). The current paper further expands the hierarchical model of Loeber et al. (in press) by demonstrating that in the majority of APD cases prior CD is a necessary precursor. The results also clarify what the psychopathological characteristics are that distinguish those CD males who progress to APD and those who do not. Logistic regression analyses demonstrate that those CD cases which score high on callous/unemotional behaviour, depression and use of marijuana are at highest risk to advance to modified APD. The findings indicate that ADHD did not contribute to modified APD once these factors had been taken into account. Also, the results show that CD cases who progress to APD, compared with CD who do not progress to APD, tend to be more violent.

The results should be considered in the light of limitations of the study. Clinic-referred samples such as the one under study are not necessarily representative of the tail end of normal distribution, and it remains to be seen whether the results hold when they are tested in a large epidemiological sample (see, e.g., Farrington, 2000). On the other hand, initial probes into the developmental antecedents of APD can best be done in high-risk samples, because of the relative low prevalence of APD in the general population. Another limitation of the current study is that the demonstrated continuity between CD and APD through callous/unemotional behaviour may only represent a key feature of antisocial development, which is evident both early

and later in the life of some youth. There is no doubt that future research will further clarify this important issue. Also, the current results will only become more solid if they are replicated in other studies.

Despite these reservations, we pose that the results may have several implications for clinical practice. We conceptualize that in CD males, depressed mood and mood-altering drugs such as marijuana may further lead to disinhibition and lack of control of normal functioning associated with APD. These features can be recorded by clinicians and help them to devise more strategies to prevent APD in CD males. There is a high need to develop experimental studies that attempt to modify these risk factors so that APD can be prevented. However, the most optimal prevention of APD must surely lie in, first, the prevention of ODD and, second, the prevention of CD. Finally, we recommend that in a future revision of the DSM-IV criteria for CD, researchers and clinicians may want to consider including callous/unemotional symptoms more explicitly in the symptom list for CD.

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